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Briefly about Dna Viruses

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Abstract

A virus is defined by its use of DNA as genetic material and relies on DNA-dependent DNA polymerase during its replication process. The genetic material in these viruses can be either double-stranded (dsDNA) or single-stranded (ssDNA). According to the Baltimore classification system, DNA viruses fall into Class I (dsDNA viruses) or Class II (ssDNA viruses). For most of these viruses to replicate, they must enter the host nucleus since they depend on the host cell's DNA polymerases to replicate their viral genome.

Keywords: DNA, RNA, Genes, DC's, Health

1. Introduction

There are two types of immunity in our body: innate immunity and adaptive immunity [1]. The field of vaccinology focuses on the adaptive immunity we acquire. This type of immunity gets activated when a pathogen successfully bypasses our innate immune system. Adapted immunity is categorized into humoral and cell-mediated immunity. Humoral immunity is associated with B cells, while T cells are linked to cell-mediated immunity. The antibodies from humoral immunity can be passed through serum containing these antibodies.

An effective immune response includes three components: (1) activation of antigen-presenting cells (APCs), (2) activation of B cells that come from bone marrow, and (3) activation of T cells that originate from the thymus. Our body's dendritic cells (specifically Langerhans cells in the skin) act as powerful APCs. They first capture the antigen and then display it on the cell surface for T-cell and B-cell receptor recognition. When immunoglobulin receptors identify foreign antigens, plasma cells are activated. These plasma cells produce different subclasses of antibodies (IgG, IgM, IgA, IgE, IgD). This exposure gives rise to memory B cells, which quickly recognize the pathogens if the body encounters them again. Memory B cells go through class-switching and somatic hypermutations to establish long-term memory and develop specific antibodies against pathogens.

2. Human Viruses

Historically, viruses have been left out of the taxonomic classification systems that apply to other living organisms [2]. This is mainly because viruses lack the key traits that define living things, like respiration, growth, reproduction, and response to stimuli. However, virologists have created a classification system based on the virus's physical features: these include the size, shape, and symmetry of the capsid, the characteristics of the envelope, and the structure of the genome. Viruses are categorized by order, family, subfamily, and genus, with the endings virales, virinae, viridae, and virus, respectively. It's important to note that not every virus receives an order or subfamily designation, as grouping by family and genus is often sufficient.

Viruses can be categorized based on the kind of genome they possess, which is particularly helpful when discussing those that infect humans. The nucleic acid found in viruses can be single-stranded (ss)DNA, double-stranded (ds)DNA, single-stranded (ss) RNA, or double-stranded (ds)RNA. Essentially, viruses can then be sorted into two main types: DNA viruses or RNA viruses. A crucial function of viral nucleic acid is to create mRNA that facilitates protein production. Nonetheless, different methods are employed by viruses to synthesize mRNA, depending on their genomic makeup. Moreover, whether a viral genome is ssRNA, dsRNA, or DNA plays a key role in activating the immune response. For example, ssRNA and dsRNA are strong triggers for inflammation through their detection by Toll-like receptors (TLR).

The level of genetic diversity seen in current virus strains reveals how long it has been since they last shared a common ancestor

and how quickly they evolve. Recombination and reassortment between different strains can further affect how this diversity appears. Several factors influence the time since the last common ancestor (LCA). For viruses that recently jumped from another species and spread swiftly among humans, the LCA likely occurred soon after this cross-species transfer. However, a single virus 'species' might encompass numerous transmission occurrences, meaning the overall diversity showcases earlier variations in the prior host. For viruses that have been infecting humans for an extended period, the duration since the LCA will likely involve population genetics processes like random genetic drift or natural selection. With random genetic drift, a chance event can cause one virus to become the ancestor of the entire species later on. This tends to happen more quickly when the effective population size (Ne), which in this case refers to the approximate long-term count of infected individuals who contribute to the next round of infections, is smaller. Conversely, when a beneficial mutation occurs, it is likely to spread quickly through the viral population, reducing much of the current genetic diversity. Both random genetic drift and a selective sweep will have a limited impact if the host population is divided.

Therefore, when we look at the current diversity of a virus, we might consider what influences have determined the 'coalescence time' or the duration back to the LCA [3]. In an idealized population without structure, the anticipated coalescence time under random genetic drift is 2Ne generations (with 'generation' here representing the average time between hosts in a transmission chain). A selective sweep, however, shortens this coalescence time, indicating that the LCA was more recent and corresponds to the last sweep that took place. If the host population remains divided, the coalescence time could extend because different viral lineages may persist in various subsets of the host population.

The mutation rate of a virus mainly determines how fast it evolves, which corresponds to the likelihood of mutations occurring during each replication and how often replication happens over time. RNA polymerases generally make more mistakes than DNA polymerases, leading to RNA viruses having higher mutation rates compared to DNA viruses. According to population genetics, the speed of neutral evolution aligns with the mutation rate. Substitutions that do not change the protein produced, known as synonymous nucleotide substitutions, tend to be neutral and thus indicate the mutation rate. On the other hand, the rates of nonsynonymous substitutions are often lowered by selective pressures on protein sequences, although they can rise due to adaptive changes; the impact of these selective influences can differ across proteins. For these reasons, comparing synonymous nucleotide substitution rates among viruses is particularly insightful, as they reveal a fundamental evolution rate.

When it comes to rapidly evolving RNA viruses, we can estimate the rate of evolution by looking at strains collected at various intervals. This rate serves as a "molecular clock," helping to identify when ancestral viruses, like the last common ancestor, existed. Although these rates can seem less consistent among various strains compared to what a random model would suggest, they are still precise enough for dating.

The level of data available on the evolution and diversity of viruses is quite varied. In this discussion, we will focus on human viruses from four well-researched families. We'll begin by exploring the origins of these viruses that infect humans and then look at how evolutionary dynamics have shaped genetic diversity since those origins.

3. Genes

The transcription of genes in DNA viruses is typically organized and regulated through a systematic cascade [4]. Immediate-early genes of adenovirus and herpesvirus activate the promoters for early genes in a specific sequence, while poxvirus particles already contain all necessary factors for early-gene transcription. Smaller DNA viruses rely less on transactivators found in their genomes for initiating early-gene transcription. Most early genes produce proteins essential for the synthesis of viral DNA and for activating late-gene transcription. The late genes mainly code for structural proteins or other proteins required for assembling the virus and its release from the infected cell. Late-gene transcription relies continuously on DNA replication, meaning that any inhibitors of DNA replication will also halt late-gene transcription.

Each DNA virus family has distinct methods for replicating their DNA. Herpesvirus DNAs exist as linear forms within the virion but become circular once inside the infected cell. During a lytic virus infection, the circular herpesvirus genomes turn into linear concatemers through a "rolling-circle" process. For viral DNA replication, herpesviruses produce a DNA polymerase along with at least six other viral proteins, and they also create extra enzymes that enhance the supply of precursor deoxynucleotide triphosphates. In adenoviruses, the genomes are linear in the virion and are copied into complementary linear versions by a virus-sourced DNA polymerase and a complex of initiator proteins. The circular double-strand genomes of papovaviruses replicate into new circular DNA molecules thanks to cellular DNA replication enzymes. Two early proteins produced by these viruses aid in both the replication of viral DNA and the lasting presence of papovavirus DNA in cells that are latently infected. The early proteins from papovaviruses encourage cells to stay in the cell cycle, which helps with viral DNA replication.

4. Dc's

Physical and chemical barriers protect the human body from viral infections [5]. However, when these defenses fail, various parts of the immune system spring into action. The immune response comprises the innate immune system, which involves Dendritic cells (DCs), Natural Killer (NK) cells, monocytes, and type I interferons (IFNs), and the adaptive immune response carried out by neutralizing antibodies and T cells. A complex interaction between various immune cell types and soluble factors from both innate and adaptive systems helps to prevent and manage chronic viral infections. Specific elements of the antiviral innate immune response include NK cells, complement proteins, and

cytokines, with DCs functioning as both immunosurveillance and immunostimulatory cells that process and present viral antigens to T cells. There are two main types of dendritic cells: plasmacytoid and conventional. Plasmacytoid DCs react to viral DNA and RNA, leading to the production of type I IFN, which stops viral replication in both infected and healthy cells. Conventional DCs are among the first immune cells to encounter viruses at the entry points into the host. The three subsets of conventional dendritic cells that play crucial roles in viral infections consist of tissue-derived migratory DCs, lymphoid-resident DCs, and monocyte-derived DCs. Tissue-derived migratory DCs include Langerhans' cells and dermal or interstitial DCs, which monitor the skin and mucoepithelial areas. Lymphoid-resident DCs exist as immature forms within the lymph nodes, spleen, and thymus. Monocyte-derived or inflammatory DCs are generated from monocytes during inflammatory responses.

Dendritic cells are strategically positioned throughout the body to effectively carry out their roles in monitoring the immune system and stimulating responses, aiming to activate both the innate and adaptive immune responses when viruses manage to overcome the body's physical and chemical defenses. On the surface of dendritic cells, various receptors like Siglec-1 (CD169), DC-SIGN, the mannose receptor, Langerin, the immune dendritic cell receptor (DCIR), heparan sulfate proteoglycan, FC gamma receptors, and syndecan-3 help with the uptake of viruses. When viruses interact with these cells, they can be broken down inside to form antigen-MHC complexes that can then be presented to T cells, prompting a T cell-mediated antiviral response. Viral antigens can originate from the replication of the virus within infected cells or from the detection of viral elements from other infected cells. Certain viruses can attach to and multiply in dendritic cells, turning them into permissive cells that help spread the virus to adjacent cells or tissues in the host. This leads to the trans-infection of lymphocytes in nearby lymph nodes. Such infections are frequently observed when viral antigens are not bound to MHC molecules, serving another mechanism to signal the immune system and stimulate T cells.

5. Immunization

Through immunization, we can trigger both active and passive immunity [1]. Generally, vaccinology focuses on active immunity. The goal of active immunization is to generate primary antibodies within the host by stimulating B-cell growth, triggering antibody production, and activating T-cells. When the body encounters the pathogen again, B cells are intensely activated during the secondary response, helping to stop the pathogen from causing more harm. Typically, active immunization offers lifelong protection. On the other hand, passive immunization involves introducing globulin products into the body, but this only offers temporary protection. It is not advised for healthy people.

Evaluating how a person responds after vaccination is crucial. Insufficient response to immunization is vital for diagnosing several primary immunodeficiencies, including common variable immunodeficiency and Wiskott-Aldrich syndrome. A faulty immune response also characterizes secondary immunodeficiencies associated with the human immunodeficiency virus (HIV).

When measuring the titer of a vaccine, it's important to focus solely on IgG responses. However, both infections and vaccinations can stimulate other types of immunoglobulins. Pre- and post-vaccination titers should be spaced at least a month apart to prevent low post-vaccination readings. For patients who received gamma globulin replacement therapy in the last eight months or those who had single doses of immune globulins like the hepatitis A vaccine in the past three to five months, a reliable immune response evaluation post-vaccination cannot be accurately obtained.

6. Availability

Vaccines can be univalent (monovalent) or polyvalent (multivalent) [1] . A monovalent vaccine is meant to protect against just one antigen or microorganism, while multivalent vaccines guard against two or more strains of the same organism or multiple different microorganisms.

The majority of vaccines on the market are either attenuated or inactivated. Some vaccines are synthetic, made using peptides, carbohydrates, or antigens. Usually, adding an adjuvant enhances the vaccine's ability to provoke an immune response. One commonly used adjuvant is alum, which boosts antibody production by slowing down the release of antigens, as seen in hepatitis B vaccines. Common types of vaccines include (i) those with live attenuated viruses; (ii) those with inactivated microorganisms; (iii) subunit vaccines; and (iv) toxoid-based vaccines. Recently, scientists have also created conjugate, recombinant, and DNA vaccines.

Live attenuated vaccines are formulated so that they do not replicate within a human host. Instead, they provoke an immune response without causing disease. They can generate a strong CD8+ cell response. Currently, live attenuated viral vaccines are used for diseases such as polio, smallpox, measles, mumps, rubella, varicella, herpes zoster, influenza, and rotavirus.

Inactivated vaccines are produced through the treatment of viruses with chemicals and heat. While these vaccines do not cause disease in the human body, they retain some unaltered epitopes that can spurt an immune response. Vaccines for the prevention of hepatitis A, hepatitis B, and rabies serve as examples of inactivated vaccines.

Subunit vaccines use parts of a pathogen rather than the whole organism to create immunity; for instance, the surface antigen from hepatitis B is utilized to develop its vaccine. In the case of toxoid vaccines, scientists incorporate inactivated toxic substances to produce the vaccines. Common examples of toxoid-based vaccines are the ones for tetanus and diphtheria.

Conjugate vaccines consist of polysaccharide antigens linked with proteins that trigger an immune response. They work well for children since their immune systems cannot respond effectively to

polysaccharide antigens by themselves. DNA vaccines emerged in 1982, where antigens from other organisms are encoded using the vaccinia virus. Generally, viruses that have large genomes serve as good candidates for this purpose. This makes it easier to replace a foreign gene in a complex viral genome. There has also been progress in inserting DNA directly into the human body, which falls under the category of DNA vaccines.

7. Hsct

A specific example of an IEI (immunological error) is SCID (Severe Combined Immunodeficiency), where managing infections is crucial both before and after HSCT (hematopoietic stem cell transplantation) [6]. Infections prior to HSCT can lead to poorer outcomes. For SCID patients who receive a transplant before infections begin, the five-year survival rate is between 80% and 95%. Thanks to successful newborn screening programs, fewer newborns with SCID are diagnosed due to infections. Still, many affected infants continue to face infections before HSCT, risking organ damage. Careful use of supportive therapies, including early prophylaxis with Bactrim and azoles, has reduced pre-HSCT infections like Pneumocystis Jirovecci pneumonia (PJP) and Candida. Controlling double-stranded DNA viruses like cytomegalovirus (CMV) is crucial before HSCT. Moreover, the use of conditioning treatment for SCID patients who have active infections is linked to poorer overall survival, which needs careful thought. After HSCT, immune reconstitution is also significant in SCID cases. For example, patients who do not have strong B cell reconstitution may suffer from low immunoglobulin levels and issues with producing specific antibodies, resulting in a heightened risk of severe bacterial infections without immunoglobulin supplementation.

Infections before undergoing HSCT are crucial to consider, especially for patients with other IEIs, as many may arrive at HSCT experiencing a high level of infections that need careful diagnosis and treatment. For instance, fungal infections significantly affect individuals with phagocytic defects. Viral infections from herpesviruses like CMV and bacterial infections can pose challenges for patients who have T cell deficiencies. To lower the chances of complications, it is beneficial to treat colonizing bacteria with antibiotics before and during HSCT, particularly for patients dealing with eczema and Staphylococcus aureus, or those facing serious lung issues with organisms like non-tuberculous mycobacteria. Patients at high risk for CMV might find prophylactic treatment with new options like letermovir advantageous. Those with phagocytic defects, like chronic granulomatous disease (CGD), can still receive HSCT even if they have active infections, including fungal ones, although using granulocyte transfusions may be necessary during periods of neutropenia.

After HSCT, infections remain a significant concern for patients with IEIs. For example, the risk of severe bacterial infections in those with hypogammaglobulinemia may be alleviated through immunoglobulin therapy. Reactivation of double-stranded DNA viruses can be addressed with antiviral drugs such as ganciclovir and foscarnet. Other viral infections, like Epstein-Barr virus (EBV) and the related lymphoproliferative disease that may arise post-HSCT, might necessitate treatments like adoptive cellular therapies involving EBV cytotoxic T lymphocytes.

8. Conclusion

A DNA virus is defined as a virus featuring a genome composed of deoxyribonucleic acid (DNA) replicated by a DNA polymerase. These viruses are categorized into two groups based on their DNA structure: double-stranded DNA (dsDNA) viruses that contain two DNA strands and single-stranded DNA (ssDNA) viruses with one DNA strand. Most dsDNA viruses fall under two realms: Duplodnaviria and Varidnaviria, while ssDNA viruses are mostly associated with the realm Monodnaviria, which includes some dsDNA viruses as well. Importantly, a number of DNA viruses do not belong to any higher taxonomic categories. Additionally, reverse transcribing viruses, which possess a DNA genome replicated through an RNA intermediate using reverse transcriptase, are classified in the kingdom Pararnavirae within the realm Riboviria.

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